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Case reports

Herpetic bronchitis with a bronchooesophageal fistula

David P Remy, Truvor V Kuzmowych, Prashant K Rohatgi, Louis G Ortega

Tracheobronchitis and oesophagitis due to herpes simplex virus (HSV) are rare. Tracheo-oesophageal fistula due to HSV oesophagitis has been described in the immunocompromised host. A case is reported of a broncho-oesophageal fistula which developed secondary to herpetic bronchitis in an apparently immuno-

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Immunosuppressed, and occasionally immunocompetent, patients are susceptible to infection of the respiratory epithelium and the gastrointestinal epithelium with the herpes simplex virus (HSV). The endoscopic changes seen in association with HSV infection of the tracheobronchial tree and oesophagus are varied, ranging from normal to areas of ulceration with or without an inflammatory membrane. This membrane consists of fibrin and a purulent exudate. The inflammation can extend deeper and produce a fistula between the tracheobronchial tree and the oesophagus. Two cases of tracheo-oesophageal fistula in association with HSV oesophagitis in im-

Abstract

competent patient.

Service, Veterans' Affairs Medical Center, Washington And Department of Pathology, George Washington University Medical Center, Washington, DC, USA L C Ortega

Pulmonary

Section, Medical Service, Veterans' Affairs Medical

Medical Center,

D P Remy T V Kuzmowych

P K Rohatgi

Laboratory

Center, Washington

and Department of Medicine, George Washington University

Washington, DC, USA

Reprint requests to: Dr D P Remy, Pulmonary Section, Medical Service, Veterans' Affairs Medical Center, 50 Irving Street NW, Washington DC 20422, USA.

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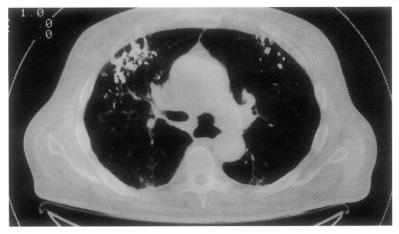


Figure 1 Computed tomographic scan of the chest at the level of the carina showing communication between the oesophagus and left main bronchus and aspirated barium in the anterior portions of the lungs.

munocompromised patients have been described. 12 Here we describe such an occurrence in an elderly, apparently immunocompetent patient.

Case report

An 82 year old man presented with a 10 day history of cough, shortness of breath, dysphagia, and vomiting. In the past he had suffered from atrial fibrillation, pernicious anaemia, and glaucoma. Medications included digoxin, procainamide, aspirin, timoptic and phosphoiodine eyedrops, and monthly vitamin B₁₂ injections. He had a 60 pack-year history of cigarette smoking but had stopped at the age of 50. He did not drink alcohol.

On examination the pulse was 84 and regular, the blood pressure was 110/60, respiratory rate 16, and temperature 37.5°C. Severe osteoarthritic changes were present in the knees. Visual acuity was poor and he was clinically dehydrated. Examination of the chest revealed diffuse rhonchi and wheezes. The cardiac, abdominal, and neurological examinations were normal. Stool was negative for occult blood.

The WBC count was 7700/mm³, platelet count 406 000/mm³, and haemoglobin 11·3 g/ dl. The peripheral blood helper T cell (CD4) count was 812/mm³ and serum electrolyte levels were normal. Digoxin and procainamide levels were therapeutic. The serum urea concentration was 61 mg/dl and the creatinine concentration was 2·2 mg/dl; these became normal after hydration. Chest radiography showed bilateral lower lobe infiltrates; abdominal radiographs were unremarkable. An ECG showed sinus rhythm rate of 100 and ST changes consistent with a digoxin effect. Arterial blood gas tensions while breathing room air showed a pH of 7.40, Paco₂ 4.8 kPa, and Pao₂ 9·2 kPa. Sputum cultures grew Staphylococcus aureus and Pseudomonas aeruginosa.

The patient was given antibiotics and intravenous fluids. All oral feedings were withheld. A barium swallow showed barium from the proximal oesophagus entering the left main bronchus, producing a bronchogram. Oesophagoscopy showed a fistula into the tracheobronchial tree at 30 cm with no other abnormalities. A computed tomographic scan of the chest confirmed a fistula between the left main bronchus and the oesophagus (fig 1). Bronchoscopic examination showed a normal trachea, a markedly inflamed carina, and the fistula through which the patient's nasogastric tube was visible. The bronchial mucosa adjacent to the fistula was covered with a white, friable membrane and biopsy samples revealed extensive necrosis, purulent exudate with many Gram positive cocci, and squamous metaplasia with no evidence of malignancy. Degenerated squamous epithelial cells with nuclear changes were also present and were characteristic of

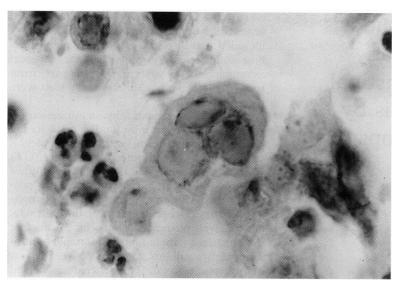


Figure 2 Biopsy specimen showing a multinucleated squamous epithelial cell with characteristic herpetic changes; note adjacent neutrophils. Stain: haematoxylin and eosin, original magnification × 40 reduced to 62% in origination.

herpetic infection (fig 2). Bronchoscopy was repeated a week later with similar biopsy findings; viral cultures grew HSV type 1. An HIV test was negative.

After 10 days of treatment with acyclovir and 21 days of antibacterial antibiotics, a third bronchoscopic examination showed considerable improvement. The patient declined insertion of endo-oesophageal prostheses or surgery to close the fistula and died on the 59th hospital day were severe aspiration pneumonia. A necropsy was not performed.

Discussion

Tracheobronchitis and oesophagitis due to HSV are well described in immunocompetent hosts.³⁻⁶ Tracheobronchitis appears to be more severe than oesophagitis which usually resolves spontaneously.3 Although the herpetic bronchitis was successfully treated with acyclovir, our patient was left with a broncho-oesophageal fistula which ultimately caused his death.

HSV can reach the lower respiratory tract by haematogenous spread or by reactivation of latent HSV in the vagus ganglion with subsequent migration along the nerves to the tracheobronchial epithelium. Most cases of herpetic tracheobronchitis, however, follow aspiration or contiguous spread from the upper airway,5 which seemed the most likely mechanism in our case.

HSV classically infects squamous epithelial cells and will also infect respiratory epithelium that has undergone squamous metaplasia. Indeed, patients with conditions that predispose to metaplasia of the respiratory epithelium (mechanical trauma, instrumentation of the airways, respiratory burns, smoking, chemotherapy, or irradiation to the lungs) have a predominance of HSV intranuclear inclusions in these metaplastic respiratory epithelial cells.46 Although metaplastic squamous epithelial cells with HSV intranuclear inclusions were seen in the bronchial biopsy samples, our patient did not seem to have any predisposing factors except perhaps the recurrent aspiration.

The first reported case of tracheo-oesophageal fistula in association with HSV infection was in a patient with leukaemia in remission.1 The fistula was caused by a primary oesophagitis from a mixed infection with herpes, aspergillus, candida, and Gram positive cocci. The second case involved a patient with HSV oesophagitis and acquired immunodeficiency syndrome.2 In our case the fistula was secondary to a severe bronchitis caused by herpes simplex in combination with Gram positive cocci. We believe this to be the first case of broncho-oesophageal fistula associated with a bronchial HSV infection in an apparently immunocompetent host.

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